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a type II Immunol. 23,						
tion of the imal lectin						
superfamily of signal-transmitting receptors. J. Exp. Med. 178, 537–547 Testi, R. et al. (1994) The CD69 receptor: a multipurpose cell-surface trigger for hematopoietic cells. Immunol. Today 15, 479–483						
Long, E.O. (1999) Regulation of immune responses through inhibitory receptors. Annu. Rev. Immunol. 17, 875–904						
Pisegna, S. et al. (2002) Src-dependent Syk activation controls CD69- mediated signaling and function on human NK cells. J. Immunol. 169, 68–74						
Zingoni, A. et al. (2000) CD69-triggered ERK activation and functions are negatively regulated by CD94/NKG2-A inhibitory receptor. Eur. J. Immunol. 30, 644–651						
Risso, A. et al. (1991) CD69 in resting and activated T lymphocytes. Its association with a GTP binding protein and biochemical requirements for its expression. J. Immunol. 146, 4105–4114						
Bikah, G. et al. (2000) Regulating T helper cell immunity through antigen responsiveness and calcium						
nains of CD69						
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Page 2 of 3 Attorney Docket No.: 27331-501 CIP2A Date of Deposit: May 18, 2007

	eposit:	May 18, 2007 Attorney Docket No.: 27331-501 CIP2A
IZSI	C10	Testi, R. et al. (1989) T cell activation via Leu-23 (CD69). J. Immunol. 143, 1123–1128
	C11	Cebrián, M. et al. (1988) Triggering of T cell proliferation through AIM, an activation inducer molecule expressed on activated human lymphocytes. J. Exp. Med. 168, 1621–1637
	C12	Santis, A.G. et al. (1992) Tumor necrosis factor-a production induced in T lymphocytes through the AIM/CD69 activation pathway. Eur. J. Immunol. 22, 1253–1259
	C13	De-Maria, R. et al. (1994) Triggering of human monocyte activation through CD69, a member of the natural killer cell gene complex family of signal transducing receptors. J. Exp. Med. 180, 1999–2004
	C14	Testi, R. et al. (1990) CD69 is expressed on platelets and mediates platelet activation and aggregation. J. Exp. Med. 172, 701–707
	C15	Ramirez, R. et al. (1996) CD69-induced monocyte apoptosis involves multiple nonredundant signaling pathways. Cell. Immunol. 172, 192–199
	C16	Walsh, G.M. et al. (1996) Ligation of CD69 induces apoptosis and cell death in human eosinophils cultured with granulocyte-macrophage colony-stimulating factor. Blood 87, 2815–2821
	C17	Cosulich, M.E. et al. (1987) Functional characterization of an antigen involved in an early step of T-cell activation. Proc. Natl. Acad. Sci. U. S. A. 84, 4205–4209
	C18	Feng, C. et al. (2002) A potential role for CD69 in thymocyte emigration. Int. Immunol. 14, 535-544
	C19	Nakayama, T. et al. (2002) The generation of mature, single-positive thymocytes in vivo is dysregulated by CD69 blockade or overexpression. J. Immunol. 168, 87–94
	C20	Lauzurica, P. et al. (2000) Phenotypic and functional characteristics of hematopoietic cell lineages in CD69-deficient mice. Blood 95, 2312–2320
	C21	Laffón, A. et al. (1991) Upregulated expression and function of VLA-4 fibronectin receptors on human activated T cells in rheumatoid arthritis. J. Clin. Invest. 88, 546–552
	C22	Remmers, E.F. et al. (1996) A genome scan localizes five non-MHC loci controlling collagen-induced arthritis in rats. Nat. Genet. 14, 82–85
	C23	Mc Indoe, R.A. et al. (1999) Localization of non-MHC collagen-induced arthritis susceptibility loci inDBA/1j mice. Proc.Natl.Acad. Sci. U. S. A. 96, 2210–2214
	C24	Brandes, M.E. et al. (1991) Transforming growth factor b1 suppresses acute and chronic arthritis in experimental animals. J. Clin. Invest. 87, 1108–1113
1	C25	Grewal, J.S. et al. (1999) Serotonin 5-HT2A receptor induces TGF-b1 expression in mesangial cells via ERK: proliferative and fibrotic signals. Am. J. Physiol. Renal Physiol. 276, F922–F930
	C26	Gorelik, L. and Flavell, R.A. (2001) Immune-mediated eradication of tumors through the blockade of transforming growth factor-b signaling in T cells. Nat. Med. 7, 1118–1122
	C27	Gorelik, L. et al. (2002) Mechanism of transforming growth factor b-induced inhibition of T helper type differentiation. J. Exp. Med. 195, 1499–1505
	C28	Cazac, B.B. and Roes, J. (2000) TGF-b receptor controls B cell responsiveness and induction of IgA in vivo. Immunity 13, 443–451
	C29	Fava, R.A. et al. (1991) Transforming growth factor 1 induced neutrophil recruitment to synovial tissues implications for TGF-b driven synovial inflammation and hyperplasia. J. Exp. Med. 173, 1121–1132
	C30	Sancho, D. et al. (1999) Activation of peripheral blood T cells by interaction and migration through endothelium: role of lymphocyte function antigen-1/intercellular adhesion molecule-1 and interleukin-1. Blood 93, 886–896
	C31	Fava, R. et al. (1989) Active and latent forms of transforming growth factor b activity in synovial effusions. J. Exp. Med. 169, 291–296
	C32	Yu, X. et al. (2001) Anti-CD69 autoantibodies cross-react with low density lipoprotein receptor-related protein 2 in systemic autoimmune diseases. J. Immunol. 166, 1360–1369
	C33	Kulkarni, A.B. et al. (1993) Transforming growth factor b1 null mutation in mice causes excessive inflammatory response and early death. Proc. Natl. Acad. Sci. U. S. A. 90, 770–774
	C34	Shull, M.M. et al. (1992) Targeted disruption of the mouse transforming growth factor-b1 gene results i multifocal inflammatory disease. Nature 359, 693–699
$\Lambda\!\!\!\!/$	C35	Shevach, E.M. (2002) CD4 ⁺ CD25 ⁺ suppressor T-cells: more questions than answers. Nat. Rev. Immuno

Page 3 of 3

Date of Deposit: May 18, 2007

Attorney Docket No.: 27331-501 CIP2A

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	2, 389–400				
C36	Chatenoud, L. et al. (1997) Induced dominant self-tolerance in overtly diabetic NOD mice. J. Immunol.				
	158, 2947–2954				
C37	Ishikawa, S. et al. (1998) A subset of CD4C T cells expressing early activation antigen CD69 in murine				
	lupus: possible abnormal regulatory role for cytokine imbalance. J. Immunol. 161, 1267–1273				
C38	Portales-Perez, D. et al. (1997) Abnormalities in CD69 expression, cytosolic pH and Ca2C during				
	activation of lymphocytes from patients with systemic lupus crythematosus. Lupus 6, 48–56				
C39	Hernández-García, C. et al. (1996) The CD69 activation pathway in rheumatoid arthritis synovial fluid T				
	cells. Arthritis Rheum. 39, 1277–1286				
C40 McGuirk, P. and Mills, L. (2002) Pathogen-specific regulatory T cells provoke a shift in the Th1/Th2					
	paradigm in immunity to infectious diseases. Trends Immunol. 23, 450–455				
C41 Swat, W. et al. (1993) CD69 expression during selection and maturation of CD4C8C thymocytes. Ex					
	Immunol. 23, 739–746				
C42	Bendelac, A. et al. (1992) Activation events during thymic selection. J. Exp. Med. 175, 731-742				
C43	Yamashita, I. et al. (1993) CD69 cell surface expression identifies developing thymocytes which audition				
	for T cell antigen receptor mediated positive selection. Int. Immunol. 5, 1139–1150				
C44	Hare, K.J. et al. (1999) CD69 expression discriminates MHC dependent and -independent stages of				
	thymocyte positive selection. J. Immunol. 162, 3978–3983				
	C36 C37 C38 C39 C40 C41 C42 C43				

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